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Nine unanesthetized, chronically instrumented, female miniature swine (MS) (avg wt, 39.7 kg) were exposed to head-to-tail inertial load (+G_z) levels of +3, +5, and +7 G_z for 60 s, with and without anti-G-suit inflation. Venous flow (VF) was measured by an electromagnetic flow sensor around the inferior thoracic vena cava at the diaphragm. Central venous pressure (CVP), abdominal venous pressure (AVP), eye-level blood pressure (ELBP), and esophageal pressure (EP) were also measured before, during, and after +G_z. There was a progressive significant decreased from control of both ELBP ($P < 0.001$) and VF ($P < 0.05$) during the three +G_z exposures, both with and without G-suit inflation. Without G-suit inflation, most of the MS were unable to tolerate +5 and +7 G. Although VF was significantly ($P < 0.02$) improved by G-suit inflation during +G_z, there was no significant difference in VF between the three +G_z levels, with or without G-suit inflation. The MS does a spontaneous straining maneuver (cyclic Valsalva) during +G_z with G-suit support. Using EP as a trigger, the data were grouped as strain or no strain (relaxation). A continuous AVP-to-CVP

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gradient existed during G-suit inflation, which increased dramatically during no strain with increasing $+G_z$, and was associated with an increase in VF. Thus, the majority of VF occurred during relaxation between strains, even though relaxation time was shortened as $+G_z$ increased. Although ELBP is obviously dependent on cardiac output and venous return, the progressive reduction in ELBP with increased $+G_z$ loads was not significantly related to changes in VF at the diaphragm which was maintained, although at a reduced rate, by the AVFP-to-CVP gradient during G-suit inflation.

Hemodynamics of miniature swine during $+G_z$ stress with and without anti-G support

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BURNS, JOHN W., MICHAEL J. PARNELL, AND RUSSELL R. BURTON. *Hemodynamics of miniature swine during $+G_z$ stress with and without anti-G support.* J. Appl. Physiol. 60(5): 1628–1637, 1986.—Nine unanesthetized, chronically instrumented, female miniature swine (MS) (avg wt, 39.7 kg) were exposed to head-to-tail inertial load ($+G_z$) levels of +3, +5, and +7 G_z for 60 s, with and without anti-G-suit inflation. Venous flow (VF) was measured by an electromagnetic flow sensor around the inferior thoracic vena cava at the diaphragm. Central venous pressure (CVP), abdominal venous pressure (AVP), eye-level blood pressure (ELBP), and esophageal pressure (EP) were also measured before, during, and after $+G_z$. There was a progressive significant decrease from control of both ELBP ($P < 0.001$) and VF ($P < 0.05$) during the three $+G_z$ exposures, both with and without G-suit inflation. Without G-suit inflation, most of the MS were unable to tolerate +5 and +7 G_z. Although VF was significantly ($P < 0.02$) improved by G-suit inflation during $+G_z$ there was no significant difference in VF between the three $+G_z$ levels, with or without G-suit inflation. The MS does a spontaneous straining maneuver (cyclic Valsalva) during $+G_z$ with G-suit support. Using EP as a trigger, the data were grouped as strain or no strain (relaxation). A continuous AVP-to-CVP gradient existed during G-suit inflation, which increased dramatically during no strain with increasing $+G_z$, and was associated with an increase in VF. Thus, the majority of VF occurred during relaxation between strains, even though relaxation time was shortened as $+G_z$ increased. Although ELBP is obviously dependent on cardiac output and venous return, the progressive reduction in ELBP with increased $+G_z$ loads was not significantly related to changes in VF at the diaphragm which was maintained, although at a reduced rate, by the AVP-to-CVP gradient during G-suit inflation.

acceleration; venous return; hydrostatic column; eye-to-heart distance; abdominal venous pressure-to-central venous pressure gradient; anti-G-suit; straining maneuver

HEAD-LEVEL ARTERIAL HYPOTENSION resulting from inertial loading in the head-to-foot vector ($+G_z$) is a common occurrence in highly maneuverable aircraft. For every +1 G_z increase in inertial load, there is a 20–25 mmHg decrease in blood pressure at head level in a relaxed seated subject. Loss of vision and loss of consciousness (LOC) are the two most distinct and potentially dangerous symptoms of $+G_z$-induced arterial hypotension. The anti-G-suit and the anti-G straining maneuver (AGSM) are the principal means of physiological protection against $+G_z$ stress. In man, the G-suit provides a reproducible increase in G-tolerance of ~2 G over mean relaxed tolerance without the G-suit of 3.7 ± 0.1 G (16). Whereas the AGSM (modified Valsalva) can increase G-tolerance, through augmentation of blood pressure, by an additional 3–4 G, depending on the physical condition of the subject and the effectiveness of the AGSM. Thus if mean blood pressure were augmented by 100 mmHg by the AGSM, then an approximate 4-G increase in G-tolerance could be expected.

Development of improved protective equipment and techniques for high $+G_z$ protection (+9 G_z) requires a basic understanding of not only the mechanisms of arterial pressure generation, but also the dynamics of venous return and the interrelationship between pressure augmentation and venous return, as influenced by G-protective equipment and techniques. Although Lindberg et al. (14) have demonstrated a 22% reduction in cardiac output in relaxed humans during +4 G_z, suggesting that venous return is compromised, the dynamics of venous return have not been adequately investigated.

Invasive hemodynamic studies in humans during $+G_z$ are difficult to justify because of inherent hazards, thus this study, using unanesthetized miniature swine, was designed to investigate the dynamic relationships between venous return, head-level arterial blood pressure, and increasing $+G_z$, as influenced by the G-suit and the AGSM.

The miniature swine has been shown to be an excellent experimental analog to man for acceleration research (2). Moreover, the unanesthetized swine performs a spontaneous, cyclic, straining maneuver during exposure to $+G_z$ while wearing an abdominal bladder G-suit; very similar to the AGSM that centrifuge subjects and pilots are trained to perform.

METHODS

Nine female miniature swine (Vita-Vet, Marion, IN), ranging in weight from 31.0 to 56.0 kg (avg wt, 39.7 kg), were chronically instrumented for experimental studies. To minimize the time that catheters were in place, requiring maintenance for patency, the instrumentation procedure was accomplished in two steps. Both steps required general anesthesia as described below. The two steps were separated by an average of 3.7 wk. The animals were initially anesthetized with 10 mg/kg thiopental sodium. Following tracheal intubation, anesthesia was maintained with a combination of halothane and nitrous

oxide.

Step 1. Through a mid-sternal incision an appropriately sized (18–20 mm ID) electromagnetic flow probe (Zepeda Instruments, Seattle, WA) was placed around the thoracic inferior vena cava at the level of the diaphragm. The flow probe was sutured to the diaphragm through two Silastic "ears" previously placed on the external surface of the flow probe.

Step 2. A 1.57-mm-ID Silastic catheter was placed in the descending arch of the aorta by way of the left brachial artery; in the superior vena cava by way of the left external jugular vein; and in the abdominal vena cava by way of the femoral vein. All catheters and leads were passed subcutaneously and exteriorized at the dorsum just craniad to the shoulders. Nylon velour had been glued to the catheters and leads at the exteriorization site, prior to instrumentation, to prevent percutaneous sinus tract infection, as described previously (26). Following surgery the animals were treated with 200 mg Polyflex im, twice daily, for 7–10 days. Catheter patency was maintained by flushing 3 times/wk with heparin (1,000 U/ml). An average of 5 wk was allowed after thoracic instrumentation before centrifuge training began, followed by an average of 3.7 wk of centrifuge training 3 times/wk at +3, +5, and +7 G_z for 1 min, with and without abdominal anti-G-suit inflation.

Flow probes were calibrated in vitro using dialysis tubing and a saline gravity flow system at room temperature. All calibrations were linear and extrapolated through zero. A number of the flow probes were used in more than one animal with consistent calibration factors (avg calibration deviation, 14.2%). In vivo zero flow was determined at the end of each experiment by exposing the animal to a $+G_z$ profile of high enough magnitude (usually 8–9 $+G_z$) to result in decompensation and unconsciousness, leading to a heart rate of 50 beats/min or less and quiescense in the other measured parameters (see Fig. 2*A*). Zero flow was taken when the flow signal showed no activity for 5 s, at which time the centrifuge was stopped and the animal recovered without any observable ill effects. Zero flow determined in this manner was identical or very close to electronic zero obtained by turning off flow probe current.

At the time of data collection the nares and nasopharynx were anesthetized with topical benzocaine and a 1.4 × 7.0-cm balloon catheter was passed through the nose and placed in the stomach for monitoring gastric pressure (GP) as an indicator of intra-abdominal pressure during the AGSM and during G-suit inflation. A second, similar balloon catheter, was passed through the same route and placed in the esophagus at heart level for monitoring esophageal pressure (EP) as an indicator of intrathoracic pressure development during the AGSM. The latex rubber balloons were adhered to PE-200 polyethylene tubing with Pliobond industrial adhesive (Goodyear Tire and Rubber, Akron, OH) and silk suture. Multiple side holes were punched in the tubing in the area of the balloon prior to balloon adhesion. The balloon catheters were connected to Statham P23De transducers. The balloons were evacuated and then inflated with 1.5–2.0 ml of air; well below the volume necessary to elicit a pressure-volume response from the balloon. Eye-level blood pressure (ELBP) was monitored with a Statham P-37 transducer attached to the aortic catheter and sutured to the skin between the eyes, after local anesthesia with a subcutaneous injection of lidocaine. Central venous pressure (CVP) and abdominal venous pressure (AVP) were monitored with Statham P23De transducers mounted caudad to the level of the heart in the horizontal *Z*-axis to allow for heart and body movement caudally during $+G_z$ (see Fig. 1) and somewhat dorsal to the heart in the vertical *X*-axis. Calibrations for CVP and AVP were corrected for the vertical hydrostatic column difference between the transducers and the heart. All transducers were mounted to ensure that they did not sense either normal gravity (-1 G_x) or the $+G_z$ load.

Following instrumentation setup, data were collected from the unanesthetized animals before, during, and after randomized combinations of centrifuge exposures of +3, +5, and +7 G_z (Fig. 1) for 1 min, with and without anti-G-suit inflation. G-suit pressure was supplied by a standard Air Force spring-loaded G-valve that opened at ~2 G and supplied the abdominal G-suit with 1.5 lb./in.^{2} (psi) per G thereafter. Thus, at +7 G_z G-suit pressure was ~7.5 psi.

Data were collected on analog tape and later digitized at a sampling rate of 128 Hz. Individual parameters were averaged over the 60 s $+G_z$ profile for each $+G_z$ level for each animal and statistically compared with control and with the other $+G_z$ levels both with and without G-suit support. In addition, using EP as a trigger, the data during $+G_z$ were separated into two groups, strain (S) and no strain (NS). When EP exceeded a predetermined threshold (2–5 mmHg), straining was considered to begin and was maintained until EP fell below the trigger threshold. NS was then the time between strains. During G-suit support, all parameters were averaged over the S-period and statistically compared with the average of the NS period. S and NS data without G-suit inflation were not statistically analyzed because of the large variability between animals and the variability over the 60 s $+G_z$ profile (see Fig. 2*A*). Recovery data were collected at 30-s intervals for 2 min following the $+G_z$ exposure. Individual recovery times were compared for significant differences.

Analyses of variance were used in all statistical tests. Student's *t* tests were used to make more specific comparisons when warranted by the results of analysis of variance. All significance levels shown in this report were taken from the follow-up *t* tests unless indicated otherwise. The most conservative significance level is sometimes used to represent multiple tests within a factor.

RESULTS

G-suit effects. Figure 2 illustrates the dramatic effects of +7 G_z on the cardiovascular system of the unanesthetized MS, without G-suit inflation (*A*), and the improvement provided with G-suit inflation (*B*). Tolerance to $+G_z$ was determined as the ability to maintain a stable cardiovascular response during $+G_z$ stress (Fig. 2*B*), which became more difficult with increased $+G_z$. More-

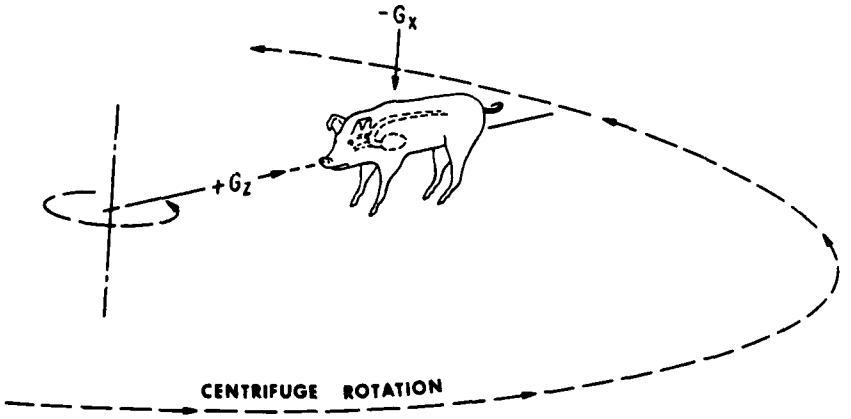

FIG. 1. Orientation of miniature swine on centrifuge. Note that +G_z inertial load was in a head-to-tail direction. −G_z is normal earth gravity. Distance from center of rotation to heart level was 13 ft.

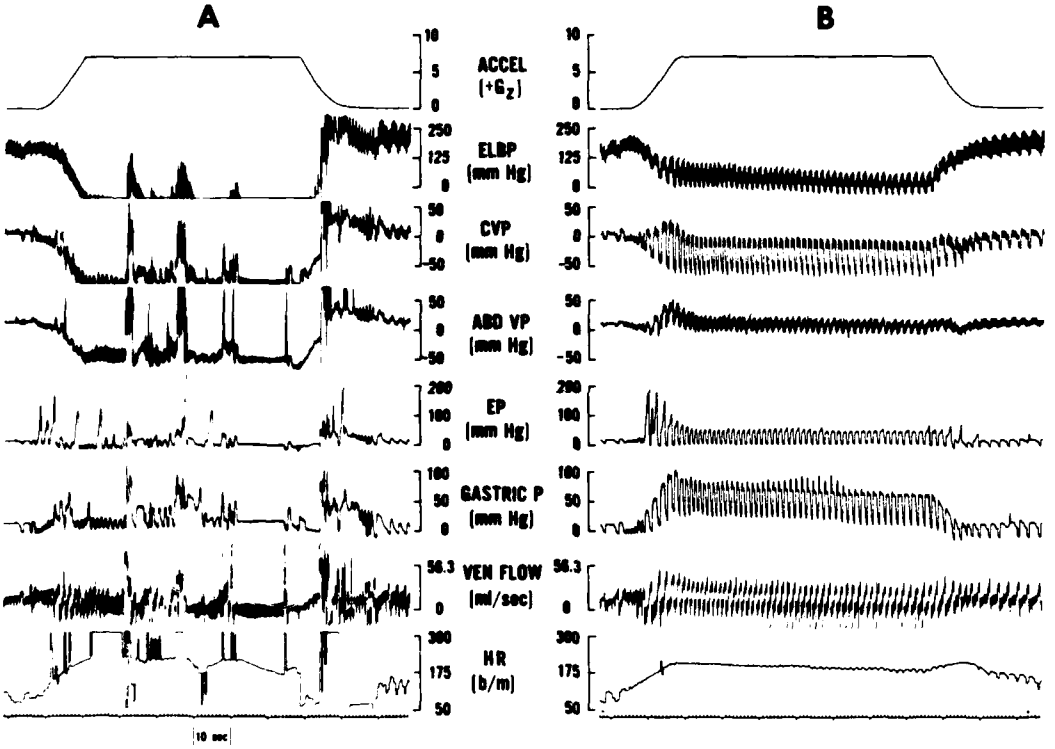

FIG. 2. *A*: acceleration exposure of miniature swine to +7 G_z for 60 s without G-suit inflation. *B*: Exposure of the same animal to +7 G_z for 60 s with G-suit inflation. ACCEL, acceleration (+G_z); ELBP, eye-level blood pressure; CVP, central venous pressure; ABD VP, abdominal venous pressure; EP, esophageal pressure; GASTRIC P, gastric pressure; HR, heart rate.

over, the benefits of the G-suit became more apparent with increased +G_z. All available data were averaged over the 60 s +G_z profile, regardless of cardiovascular stability. However, in a number of instances, data were not available for a variety of typical reasons (plugged catheters, electronics failure, etc.). Stability without G-suit inflation was observed in 7 of 8 animals at +3 G_z, 3 of 8 animals at +5 G_z, and only 1 of 8 animals at +7 G_z. Whereas stability with G-suit inflation was observed in 8 of 8 animals at +3 G_z, 8 of 8 animals at +5 G_z, and 6 of 7 animals at +7 G_z. Similar results have been previously observed (11–13, 25). Table 1 lists the mean ± SE response of the measured parameters to the three +G_z levels with and without G-suit inflation.

The heart rate (HR) data from Table 2 also demonstrate the beneficial effect of the G-suit. Although the HR response at +3 G_z was essentially the same with or without G-suit inflation, at +5 and +7 G_z without G-suit heart rate at 60 s had deteriorated (dramatic bradycardia in some animals) as loss of consciousness approached or was evident; heart rate with G-suit inflation was maintained at a more stable level throughout the +G_z exposure (Fig. 2*B*). In addition, the data at 60 s without G-suit inflation illustrate the progressive increase in degree of difficulty in maintaining cardiovascular stability with increasing +G_z.

TABLE 1. *Hemodynamic response of the miniature swine to +G_z stress*

	+3 G_z				+5 G_z				+7 G_z			
	W/OS	n	WS	n	W/OS	n	WS	n	W/OS	n	WS	n
ELBP, mmHg												
C	127.8±4.9	8	125.5±5.2	8	130.6±5.7	8	131.0±6.1	8	124.9±8.7	8	131.5±6.8	8
G	31.6±14.0	8	62.4±8.2	8	−17.3±14.5	8	46.3±10.1	8	−75.5±28.2	5	2.5±10.5	7
G(S)	35.7±15.6	7	62.9±8.7	7	−18.0±16.1	7	50.7±10.7	7	−64.3±25.4	5	9.4±12.5	6
G(NS)	17.2±15.2	7	46.6±8.0	7	−21.9±22.4	7	19.3±12.4	7	−89.9±24.5	5	−26.3±12.8	6
CVP, mmHg												
C	4.9±2.0	6	4.4±2.1	6	4.0±3.7	6	3.9±2.7	6	5.0±3.1	6	4.2±2.4	6
G	−21.0±6.6	6	−15.0±6.4	5	−33.9±7.8	6	−10.2±5.3	6	−44.3±9.8	6	−15.2±6.2	6
G(S)	−13.7±7.0	6	−9.1±6.9	5	−26.6±8.8	6	0.4±5.6	6	−34.9±9.6	6	−5.3±7.4	6
G(NS)	−30.1±4.6	6	−27.6±5.2	5	−48.7±4.7	6	−41.2±3.5	6	−59.1±8.1	6	−49.4±4.7	6
AVP, mmHg												
C	8.2±2.6	5	9.4±3.0	4	11.9±4.4	6	10.0±2.1	6	8.0±3.0	6	8.9±2.3	6
G	−11.1±7.2	5	3.1±5.9	4	−23.8±8.0	6	9.6±7.3	7	−42.8±11.0	6	10.5±9.6	7
G(S)	−8.5±6.8	5	4.6±6.2	4	−17.6±8.0	5	8.4±8.0	6	−29.5±11.5	5	7.9±9.9	6
G(NS)	−15.9±6.1	5	−0.2±4.9	4	−26.2±9.8	5	2.0±8.1	6	−42.1±9.3	5	3.5±6.4	6
EP, mmHg												
C	9.0±4.0	8	4.6±1.4	8	6.4±2.2	8	4.6±1.6	8	8.9±3.2	8	5.8±1.7	8
G	10.5±5.8	8	13.7±6.4	8	13.7±4.7	8	22.1±4.0	8	13.4±4.4	8	27.5±3.7	8
G(S)	20.3±4.8	8	19.6±4.9	8	22.7±4.4	8	30.3±4.9	8	24.8±3.9	8	35.6±4.9	8
G(NS)	−2.5±1.6	8	−3.0±1.5	8	−3.4±1.3	8	−2.1±1.4	8	−5.2±1.3	8	−2.4±1.5	8
GP, mmHg												
C	4.4±1.4	8	5.0±1.6	8	5.5±2.5	8	4.3±1.5	8	3.6±1.1	8	5.1±0.9	8
G	16.5±4.2	8	21.2±4.8	8	26.1±4.6	8	39.3±6.9	8	30.4±5.8	8	48.6±4.5	8
G(S)	22.9±3.8	8	27.2±3.8	8	28.9±6.1	8	45.7±7.3	8	35.5±7.0	8	58.7±6.3	8
G(NS)	10.5±2.5	8	13.2±6.1	8	15.6±4.4	8	20.7±8.6	7	17.2±5.6	8	13.3±4.6	8
VF, ml/s												
C	13.7±2.9	8	13.6±3.3	8	15.8±3.0	8	15.6±3.0	8	14.9±3.0	8	15.6±3.4	8
G	8.0±2.7	8	10.8±3.1	8	8.3±2.5	8	10.7±2.8	8	5.3±1.5	8	9.6±2.5	8
G(S)	−1.4±4.0	7	−0.1±3.1	7	1.8±1.8	7	2.5±2.4	7	3.6±2.8	7	2.3±2.9	7
G(NS)	18.5±7.2	7	22.8±6.0	7	18.7±8.0	7	25.8±4.2	7	15.8±5.9	7	29.1±6.3	7
G-Suit P, psi	0.0		1.8±0.2	6	0.0		4.8±0.2	7	0.0		8.0±0.3	7
%Time S	44.5±11.0	8	55.3±8.7	8	56.6±7.4	8	74.3±2.8	8	56.6±8.5	8	78.5±2.1	8
%Time NS	55.5±11.0	8	44.7±8.7	8	43.4±7.4	8	25.7±2.8	8	43.4±8.5	8	21.5±2.1	8

Data are means ± SEM. W/OS, without G-suit; WS, with G-suit; C, control before +G_z; G, mean data over 60 s +G_z exposure; S, mean data during straining maneuver; NS, mean data during no-strain (inspiration); ELBP, eye-level blood pressure; CVP, central venous pressure; AVP, abdominal venous pressure; EP, esophageal pressure; GP, gastric pressure; VF, venous flow.

TABLE 2. *Heart rate response of the miniature swine to +Gz stress*

	+3 G_z				+5 G_z				+7 G_z			
	W/OS	n	WS	n	W/OS	n	WS	n	W/OS	n	WS	n
Control	97.7±13.7	7	103.5±11.6	6	92.0±8.2	6	97.2±5.7	6	81.3±3.2	7	100.8±6.0*	6
During	215.4±8.9	7	215.0±9.8	6	221.0±9.5	6	208.3±11.8†	6	215.4±11.7	7	210.7±10.3	7
60 s	204.6±8.6	5	201.0±9.9	6	151.4±40.5	5	191.4±12.3	7	122.7±32.0‡	7	181.3±16.0†	7

Data are means ± SEM. W/OS, without G-suit; WS, with G-suit; control, heart rate (HR) before +G_z; During, peak HR during the +G_z exposure (generally occurred early in exposure); 60 s, HR at the end of the 60 s +G_z exposure. Significant difference between W/OS vs. WS. * $P < 0.01$. † $P < 0.025$. Significant difference between +3 G_z W/OS vs. +7 G_z W/OS. ‡ $P < 0.05$.

Figure 3 demonstrates the relationship between measured arterial ELBP, calculated arterial brain level and heart level blood pressure, and venous flow at the three +G_z levels, both without and with G-suit inflation. ELBP decreased from control ($P < 0.001$) by 75%, 113%, and 160%, respectively at +3, +5, and +7 G_z without G-suit inflation. Whereas ELBP was significantly ($P < 0.05$) augmented with G-suit inflation, resulting in decrements from control ($P < 0.001$) of only 50%, 65%, and 98% respectively, at the same +G_z levels. The comparison of ELBP between +G_z levels, both with and without G-suit inflation, also showed significant ($P < 0.025$) differences (Fig. 3); however, the magnitude of the ELBP reduction during +G_z was greater without G-suit inflation. The regression of ELBP reduction from control in response to +G_z, with and without G-suit inflation, illustrates this difference (Fig. 4). The correlations for both equations of Fig. 4 were highly significant ($P < 0.001$). The reduction in ELBP/G without G-suit inflation was 26.4 mmHg, whereas, with G-suit inflation it was 15.9 mmHg, a significant difference ($P < 0.025$).

Venous flow decreased from control by 41, 48, and 64% without G-suit inflation ($P < 0.025$) and by 20, 31, and 39% with G-suit inflation ($P < 0.05$) at the three respective +G_z levels (Fig. 3). There were no significant differences between venous flow at the three +G_z levels, with or without G-suit inflation, although venous flow with suit inflation was greater than without ($P < 0.05$).

CVP during +G_z was significantly ($P < 0.025$) reduced from control, both with and without G-suit inflation, but

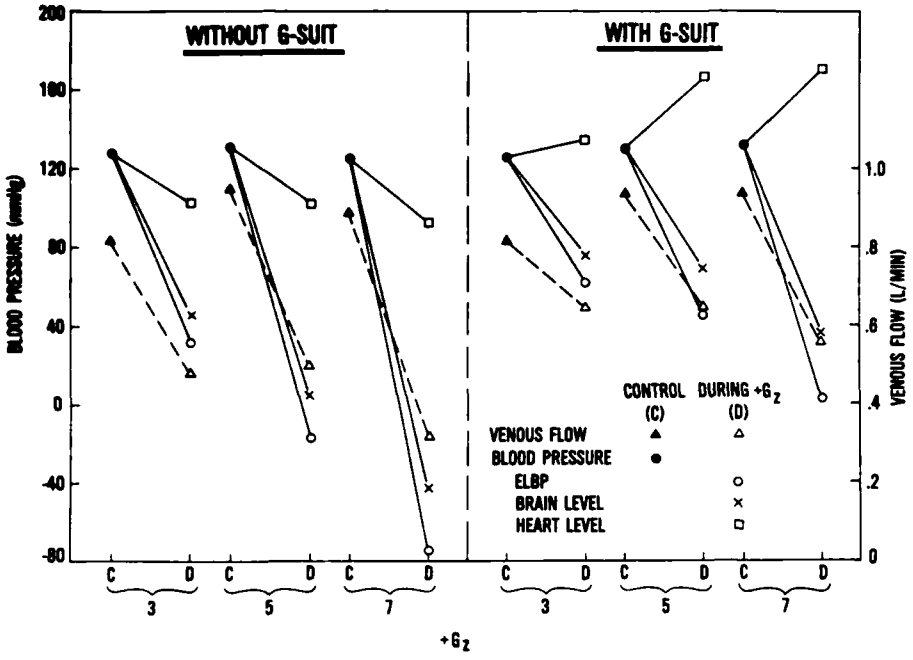

FIG. 3. Effect of +3, +5, and +7 G_z on mean data of measured eye-level blood pressure (ELBP) and venous flow, and calculated brain level and heart level blood pressure, without and with G-suit inflation. ELBP, brain level BP, and heart level BP are assumed to be equal during control. Calculations for brain level and heart level BP are discussed in text.

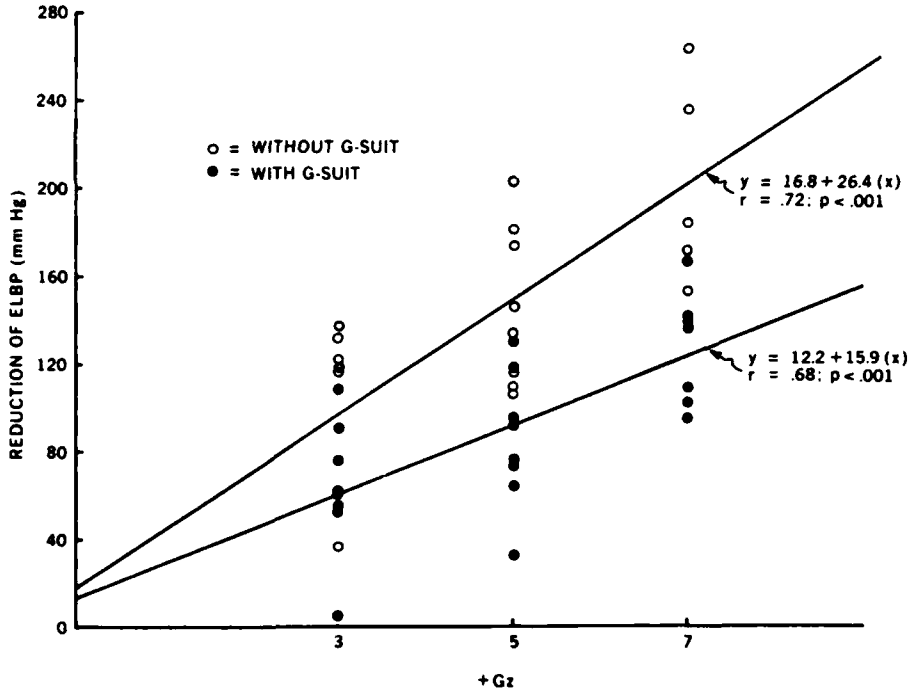

FIG. 4. Linear regression relationship between reduction of eye-level blood pressure (ELBP) from control and +G_z level. Reduction of ELBP without G-suit inflation was 26.4 mmHg/G, whereas with G-suit inflation it was 15.9 mmHg/G.

the reduction was less pronounced with G-suit inflation ($P < 0.005$). Without inflation, the reduction from control was greater with increased +G_z ($P < 0.05$), whereas with inflation the reduction remained constant. AVP decreased from control ($P < 0.005$) at +5 and +7 G_z without suit inflation but showed no change from control with inflation.

Analysis of EP showed a significant increase from control at +5 and +7 G_z with inflation ($P < 0.005$). The +7 G_z change was greater than the +3 and +5 G_z changes ($P < 0.05$). EP did not change from control without G-suit inflation. The positive EP during control (Table 1) was the result of an unusual respiratory technique during resting control. The technique was similar to the straining maneuver during +G_z, although at a much lower amplitude. A quick inspiration resulting in a negative EP was followed by breath holding and intrathoracic pressure development followed by expiration and repeat of the cycle. Breath holding with slight straining was the major part of the respiratory cycle, therefore, the average of EP over the control period was positive (Table 1). However, note that during +G_z EP was negative during

the NS part of the straining cycle at all three +G_z levels, with and without G-suit inflation.

Gastric pressure was significantly greater than control during all +G_z runs ($P < 0.025$). The changes at +5 and +7 G_z with and without G-suit inflation were greater than at +3 G_z ($P < 0.025$), but +5 and +7 G_z did not differ from each other. Moreover, the with G-suit inflation change was greater than the without G-suit inflation change at both +5 and +7 G_z ($P < 0.025$).

Straining effects. The spontaneous straining maneuver of the MS, and its influence on the measured parameters is shown in Fig. 5. Five seconds of the record was run at a faster speed to better illustrate the cyclic and rhythmical nature of the straining maneuver. Vertical dashed lines were placed at the beginning of the straining maneuver (*A*) and at the point where the animal began to relax the strain (*B*) to illustrate the relationship between EP and the other parameters, especially venous flow. Fluctuations in intrathoracic pressure, as measured by EP, were transmitted to ELBP, CVP, and GP, and to a lesser degree, AVP. The sharp increase in the EP at the beginning of the straining maneuver (*A*) was coincident with a sharp decrease in VF at the diaphragm to a negative value (Fig. 5). VF then oscillated around zero while EP was still elevated. At initiation of relaxation of the straining maneuver (*B*) VF increased, and peak flow occurred just prior to the next strain.

An AVP-to-CVP (AVP-CVP) gradient was observed at all +G_z levels, with or without G-suit inflation. The gradient was smallest during the strain and greatest during no strain (Fig. 6). Moreover, the with G-suit inflation no strain gradient increased with increased +G_z. Concurrent with the AVP-CVP gradient during +G_z, VF was lowest during the strain and greatest during no strain. Thus, the AVP-CVP gradient was augmented by the G-suit and appeared to be the driving force for VF through the diaphragm during +G_z, even against the higher +G_z inertial loads.

From Fig. 5 it can be seen that the straining maneuver, as monitored by EP, influenced nearly all of the measured parameters. It was of interest, therefore, to determine the temporal relationship between the increase in EP during straining and the increase in ELBP, CVP, and AVP over the same period. The inclusive linear regression and significant correlation of ΔEP on ΔELBP at all three +G_z levels with G-suit inflation are shown in Fig. 7. Both ΔEP and ΔELBP were determined as average pressure during strain minus average pressure during no strain. The correlations at individual +G_z levels were also significant (+3 G_z, $r = 0.88$, $P < 0.025$; +5 G_z, $r = 0.87$, $P < 0.025$; and +7 G_z, $r = 0.91$, $P < 0.001$). The inclusive correlation of ΔEP with ΔCVP at the three +G_z levels with G-suit inflation was also significant ($r = 0.84$, $P = 0.001$). There was no significant correlation of ΔEP with ΔAVP.

As anticipated, all parameters demonstrated a significant ($P < 0.05$ or less) difference between S and NS with G-suit inflation. The significant values are not included in this report; however, the S and NS data were used in the development of Figs. 6 and 7.

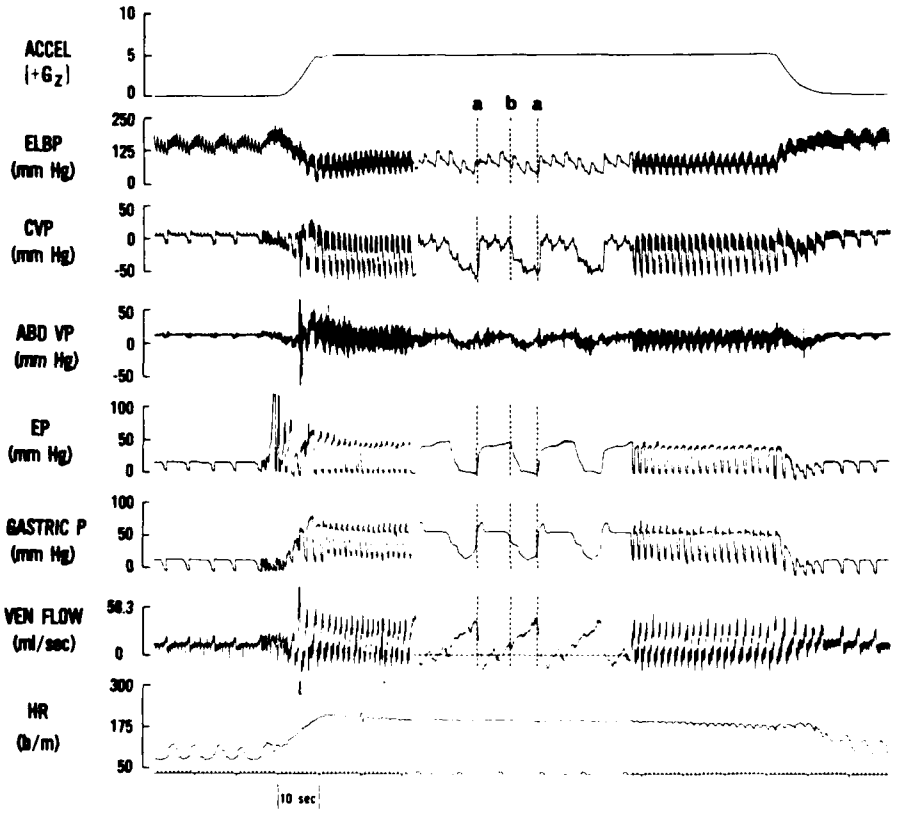

FIG. 5. Influence of the anti-G straining maneuver, measured as esophageal pressure (EP), on other measured parameters, especially venous flow. Note that at initiation of the straining maneuver (*a*) flow decreased dramatically and remained low until relaxation of the strain (*b*) when it increased and was greatest just prior to the next strain. See Fig. 2 for legend.

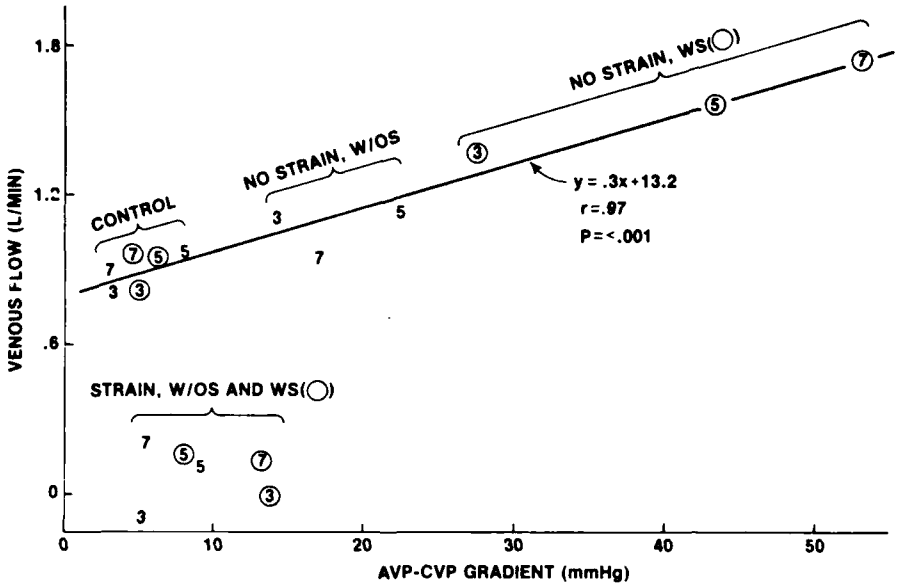

FIG. 6. Relationship between the abdominal venous pressure-central venous pressure gradient and venous flow before (control) and during the 3 +G_z levels, during strain and no-strain. Inspiration and the major part of expiration occurred during no strain. Data are shown with (WS) and without (W/OS) G-suit inflation. Circled numbers are data at that +G_z level WS. Regression equation includes control plus no strain data.

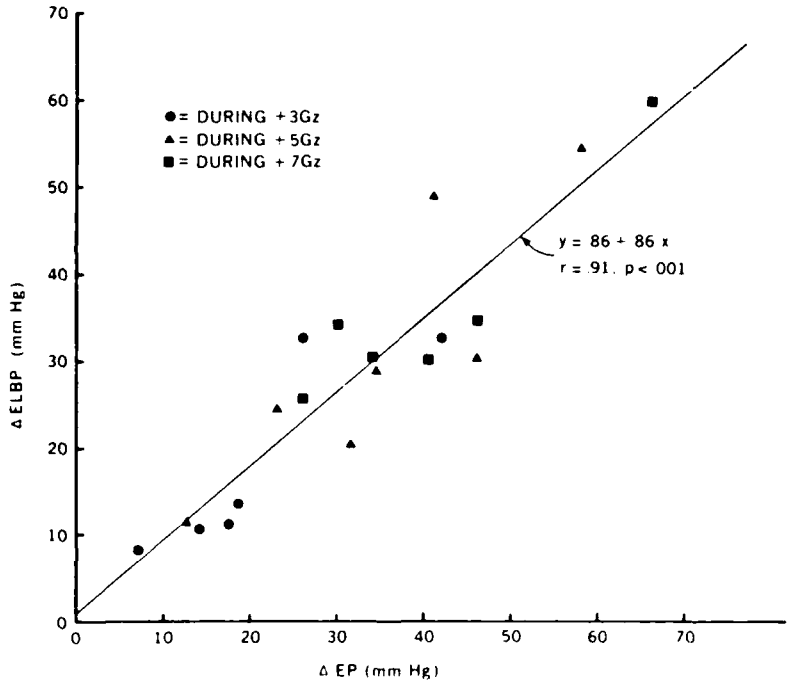

FIG. 7. Effect of the straining maneuver (EP) on eye-level blood pressure (ELBP) during the three +G_z levels with G-suit inflation. Regression equation includes data from all three +G_z exposures. EP, esophageal pressure; ΔEP and ΔELBP, difference between mean data during strain and mean data during no strain over the 60 s +G_z profile.

Statistical analysis of recovery data at 0, 30, 60, and 90 s post +G_z showed several significant but unremarkable differences. In general, all of the parameters demonstrated two phases of recovery. The first phase, which started at the offset of +G_z, was immediate and very dramatic, but opposite to the onset response to +G_z. The second phase of the recovery response was a gradual stabilization toward control levels over the following 90 s.

DISCUSSION

These data demonstrate that VF through the diaphragm occurs at all +G_z levels, with and without G-suit support (Fig. 3). Rushmer has demonstrated an abdominal-to-thoracic pressure gradient in dogs during +G_z, which he related to the hydrostatic column effect of the abdominal contents (19). He showed that during +5 G_z the pressure developed in the abdomen would support a column of water to a level above the dome of the diaphragm. Thus the pressure at any point in the abdomen was dependent on its distance below the diaphragm and the +G_z level. From Fig. 6 it can be seen that the AVP-CVP gradient at heart level during control varied between 3 and 8 mmHg before the three +G_z exposures. During +G_z, without the strain, the AVP-CVP gradient was dramatically augmented with G-suit inflation in proportion to the +G_z level and the accompanying G-

suit pressure, and was associated with increased venous flow. Whereas, without G-suit inflation, the AVP-CVP gradient and VF during NS were not nearly as great. Thus, the AVP-CVP gradient during NS was augmented by +G_z but more significantly by G-suit inflation and its effect on the hydrostatic column of the abdominal contents (19). During the straining maneuver both the AVP-CVP gradient and VF were reduced because of the increase in intrathoracic pressure (damming effect). Thus the increase in abdominal pressure that normally occurs during G-suit inflation at the higher +G_z levels was counteracted by an increase in intrathoracic pressure during S.

The progressive increase in VF with increasing +G_z during NS with G-suit inflation, as illustrated in Fig. 6, is deceptive. From Fig. 3 it can be seen that total VF was reduced at +7 G_z with or without G-suit compared with the other two +G_z levels, whereas Fig. 6 shows the greatest flow at +7 G_z during NS. An explanation is found in Table 1. Note that percent of time during NS was reduced to 21.5% of total time with G-suit inflation at +7 G_z. Therefore, total VF (Fig. 3) was not purely the algebraic sum of S and NS venous flow data. Note also in Table 1, the miniature swine maintained the AGSM longer with G-suit inflation than without.

Normally, veins above the heart are collapsed and veins below the heart are distended. It is probable that during +G_z the veins caudad to the heart are distended to a greater extent than normal by the increased +G_z load, but still supported by surrounding tissue (19). During normal respiration it is generally accepted that venous flow through the diaphragm increases during inspiration and decreases during expiration (15, 23, 24). However, there is some controversy over the mechanism of flow during the Valsalva and the Mueller maneuver and during deep inspirations. Flow through the vena cava at the diaphragm follows the principles of flow through collapsible tubes (7, 8), i.e., an increase in upstream (abdominal) pressure will cause an increase in flow through the system, whereas an increase in downstream (intrathoracic) pressure causes a reduction or stoppage of flow through the system. However, a decrease in downstream pressure does not necessarily increase flow through the system. In fact, a large decrease in downstream pressure may collapse the thoracic vena cava, stopping flow momentarily. Thus during the Mueller maneuver or during a deep inspiration there is evidence that the thoracic vena cava collapses or is reduced in diameter, thus obstructing any large surge of blood into the thorax (5, 24). In this study there was no indication of a collapse of the thoracic vena cava during rapid inspiration. In fact, VF was at its highest value during rapid inspiration (Fig. 5). Contributing factors that promoted VF during rapid inspiration during +G_z were *1*) distended capacitance vessels caudad to the heart, which acted as a ready reservoir; and 2), the large increase in the AVP-CVP gradient during NS (Fig. 6), which included expiration and rapid inspiration.

It has been reported that inferior vena cava flow in the dog is about two-thirds of total venous return to the heart (10). It was anticipated that VF, as measured in this study, would yield similar results. Measurement of resting cardiac output from several of the MS by dye dilution (Cardio-Green) resulted in a mean value of 3.25 l/min. Thus two-thirds of 3.25 l/min is 2.17 l/min. The greatest value for mean VF during control was 0.95 l/min (15.8 ml/s, Table 1), considerably less than anticipated. It is possible that in the MS the distribution of cardiac output is not the same as in the dog; a larger portion may go to areas above the heart that are drained by the superior vena cava. Moreover, the azygos vein may play a greater role in venous return in the MS. The possibility also exists of mechanical obstruction of the vena cava by the flow probe or by the development of scar tissue around the flow probe site. In fact, two animals were discarded because of ascites. At necropsy these animals showed mechanical obstruction through scar tissue development at the flow probe site. However, no gross obstruction was observed at necropsy of the MS used in this study. Moreover, mean VF was measured as high as 1.75 l/min (29.1 ml/s, Table 1) at +7 G_z during no strain, demonstrating that flow was not obstructed and was able to significantly increase from the resting control value. The observed reduction in VF from control by 20% at +3 G_z and 31% at +5 G_z with G-suit inflation compare favorably with previously reported decreases in cardiac output in MS of 22 and 45% during similar circumstances (11) and a 22% cardiac output reduction in relaxed humans at +4 G_z without G-suit inflation (14).

Although ELBP is dependent on cardiac output and venous return, it was not surprising that no statistically demonstrable cause and effect relationship was observed between the decrease in ELBP and VF during +G_z, with or without G-suit inflation due to individual animal variability and the fact that pressure was measured at eye level and VF was measured at the diaphragm. Rather, ELBP was closely related to the +G_z level (Fig. 4). Using the relationship $P = \rho gh$ (ρ, density of blood; g, acceleration units; and h, distance from eye to heart) the hydrostatic pressure (P) of a column of blood from the eye to the base of the heart was calculated for +3, +5, and +7 G_z. Thus, P is equal to the reduction of ELBP at the respective +G_z level. Previous static measurements of h from 28 adult (2 yr or older) MS ranged from 27.9 to 34.3 cm (mean = 30.9 cm). The MS ranged in weight from 40.7 to 54.6 kg (mean = 47.7 kg). No relationship was found between body weight and h, therefore the mean value of 30.9 cm was used for the above calculation of P. The range and mean weight of the nine MS used in this study were 31.0–58.6 kg and 39.6 kg, respectively. Calculated P was 72.1 mmHg at +3 G_z, 120.2 mmHg at +5 G_z, and 168.3 mmHg at +7 G_z, i.e., blood pressure at eye level would decrease by 24 mmHg for every 1 G increase in the +G_z load. Figure 4 illustrates the actual reduction in ELBP, with and without G-suit inflation, observed in this study. The calculated values of P fall between the with and without G-suit data at all three +G_z levels. There are two possible explanations for the discrepancy between the actual and calculated data: *1*) The mean value of 30.9 cm for h obtained from other MS may not be appropriate for the animals used in this study. The mean weight of these MS was 8.1 kg less than the

previous MS. However, that does not necessarily indicate that they also had a shorter *h*, since they were of the same age and there was no relationship between body weight and *h*, as noted above; and 2), without G-suit support the heart was displaced caudally during +G_z, increasing *h* and resulting in a greater reduction of ELBP, as illustrated in Fig. 4. Conversely, when the abdominal bladder G-suit inflated during +G_z the diaphragm was elevated, which in turn elevated the heart and reduced *h*, causing a decreased reduction of ELBP (Fig. 4). The latter explanation is more reasonable and fits the data very nicely. Sieker et al. (22) found very similar results in man with and without G-suit support. They reported a decrease in ELBP of 25 mmHg/G without G-suit inflation and 12.5 mmHg/G with G-suit inflation.

Loss of vision occurs when ELBP falls below intraocular pressure (20–25 mmHg). However, the ability of the MS to maintain adequate brain blood flow, and thus consciousness, at the moderately negative ELBPs observed in this study (Table 1) can be explained by several factors. *1*) The brain of the MS is ~6 cm caudad to the eye, providing about 14, 23, and 33 mmHg greater brain level arterial blood pressure at +3, +5, and +7 G_z, respectively, compared with ELBP. *2*) During +G_z, cerebrospinal fluid pressure and venous pressure craniad to the heart decrease in proportion to the +G_z load (20, 27), resulting in a negative intracranial and jugular pressure (6). *3*) The dermis of the MS is much thicker and stiffer than that of man, providing additional support against collapse of the neck arteries and veins. The negative intracranial and cerebrospinal pressures could result in passive intracranial vasodilation. Moreover, the negative jugular pressure has been postulated to create a siphon effect which passively draws blood through the brain in proportion to the arterial-venous pressure gradient and the degree of intracranial vasodilation. Measurement of O_2 saturation of blood from the jugular vein showed that it was barely affected by acceleration up to +4.5 G_z, suggesting adequate blood flow through the brain at that +G_z level (6). This hypothesis is further supported by data from Laughlin et al. (12) who measured cerebral blood flow in the MS during similar circumstances at +3, +5, and +7 G_z with G-suit inflation, using radiolabeled microspheres. They observed that an aortic blood pressure of 140 mmHg was necessary for adequate cerebral blood flow at +7 G_z. Heart level blood pressure of 140 mmHg translates to an ELBP of −28.7 mmHg in the present study. Three of the MS had an ELBP of −7.8, −6.4, and −5.4 mmHg and had stable cardiovascular responses with rhythmical straining maneuvers at +7 G_z with G-suit inflation, indicating adequate cerebral blood flow. Only one of the seven MS at +7 G_z with G-suit inflation had an unstable cardiovascular response, with an ELBP of −39.6 mmHg (calculated heart level blood pressure of 129 mmHg), suggesting an inadequate cerebral blood flow. The lowest ELBP associated with consciousness and struggling, indicative of a marginally adequate cerebral blood flow, was −60 mmHg at +7 G_z without G-suit inflation. More negative pressures (3 observations) were associated with unconsciousness and inadequate cerebral blood flow.

The anti-G-suit provided a fairly consistent amount of G-protection through a number of mechanisms: *1*) reduction of the eye-to-heart hydrostatic column by elevation of the diaphragm (19); *2*) an increase in the AVP-CVP gradient promoting venous return; and *3*) augmentation of peripheral resistance (18), thus reducing arterial run-off and venous pooling caudad to the heart. A large part of the increased peripheral resistance during +G_z results from reflex autonomic activity induced by numerous cardiopulmonary pressure and volume receptors (1, 4, 9, 17, 21). In addition, the anti-G suit provides a "platform" for the animal to strain against. Note in Fig. 2*A*, without G-suit inflation the animal does a very poor straining maneuver and thus does poorly during +G_z, whereas with G-suit inflation (Fig. 2*B*) the effectiveness of the AGSM can be observed in the other measured parameters. Even with G-suit inflation, +7 G_z is approaching the average upper limit of tolerance for the MS used in this study and in others (11, 12). The G-suit used, although very effective, does not provide G-protection below the abdomen, as do the standard U. S. Air Force and Navy G-suits (additional bilateral thigh and calf support). However, in man, inflation of the abdominal bladder alone has been shown to provide 75% of the relaxed G-protection provided by the standard 5 bladder G-suit (3, 28). Whereas, inflation of only the leg bladders provided the other 25% of protection (3). Additional G-protection might have been obtained by development of a G-suit to cover the hindlegs. However, the anatomy of the MS hindlegs precluded that possibility.

The transducers for the measurement of CVP and AVP were aligned to allow for approximate positional shift of catheter tip during +G_z; however, undocumented movement of the catheters in the chest or abdomen, especially without G-suit inflation, and movement of the whole body caudally during +G_z resulted in measurement error which became greater with increased +G_z. Displacement of the catheters from control position would result in an error of 0.78 $mmHg \cdot cm^{-1} \cdot G^{-1}$, i.e., for every 1 cm displacement of the catheters from control position there would be an error of 5.5 mmHg in CVP or AVP at +7 G_z. Without knowing the position of the catheters during +G_z it was not possible to correct for catheter displacement. However, since both the CVP and AVP transducers were in the same vertical plane the AVP-CVP gradient error would be minimized.

Neither gastric pressure nor AVP were proportionately influenced by the actual G-suit pressure. Average G-suit pressure at +3, +5, and +7 G_z was 1.8 psi (93 mmHg), 4.8 psi (248 mmHg), and 8.0 psi (414 mmHg), respectively. At the same time GP during strain, measured by a balloon in the stomach, averaged only 19.6 mmHg at +3 G_z, 45.7 mmHg at +5 G_z, and 58.7 mmHg at +7 G_z, whereas AVP showed only minor fluctuations during the AGSM (Fig. 5). Maximum measured GP during NS with G-suit inflation (G-suit only effect) was 20.7 mmHg at +5 G_z (Table 1). Therefore, it appears that intra-abdominal pressure development during G-suit inflation is buffered by diaphragm distention as the compliant abdominal contents are displaced by the G-suit. These

results are similar to previously reported observations (19).

In summary, it has been demonstrated that VF through the diaphragm occurred at all $+G_z$ levels, both without G-suit and, especially, with G-suit support. Moreover, no significant relationship existed between the decrease in VF and the decrease in ELBP during $+G_z$, i.e., there was still significant VF through the diaphragm at +7 G_z without G-suit inflation even though mean ELBP had decreased by 160% to −75.5 mmHg.

These data also indicate that G-suit pressure contributes not only directly to hemodynamics through increased peripheral resistance, but also indirectly through elevation of the diaphragm and the heart, thus increasing the abdominal hydrostatic column and the AVP-CVP gradient, as well as decreasing the eye-to-heart hydrostatic distance (h). The AVP-CVP gradient appears to be the driving force of VF through the diaphragm during $+G_z$.

The AGSM (S, Table 1) contributed directly, in a positive linear fashion, to the augmentation of ELBP (Fig. 7), while at the same time impeding VF through the diaphragm, thus demonstrating the need for an efficient, rhythmical, cyclic AGSM which generates pressure for ELBP support during the strain and allows VF through the diaphragm during relaxation (NS, Table 1).

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